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AIR POLLUTION ASPECTS OF BIOLOGICAL AEROSOLS (MICROORGANISMS)

Harold Finkelstein

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Bethesda, Maryland

September 1969

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AIR POLLUTION ASPECTS
OF
BIOLOGICAL AEROSOLS
(MICROORGANISMS)

Prepared for the
National Air Pollution Control Administration
Consumer Protection & Environmental Health Services
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FOREWORD

As the concern for air quality grows, so does the concern over the less ubiquitous but potentially harmful contaminants that are in our atmosphere. Thirty such pollutants have been identified, and available information has been summarized in a series of reports describing their sources, distribution, effects, and control technology for their abatement.

A total of 27 reports have been prepared covering the 30 pollutants. These reports were developed under contract for the National Air Pollution Control Administration (NAPCA) by Litton Systems, Inc. The complete listing is as follows:

- Aeroallergens (pollens)
- Aldehydes (includes acrolein and formaldehyde)
- Ammonia
- Arsenic and Its Compounds
- Asbestos
- Barium and Its Compounds
- Beryllium and Its Compounds
- Biological Aerosols (microorganisms)
- Boron and Its Compounds
- Cadmium and Its Compounds
- Chlorine Gas
- Chromium and Its Compounds (includes chromic acid)
- Ethylene
- Hydrochloric Acid
- Hydrogen Sulfide
- Iron and Its Compounds
- Manganese and Its Compounds
- Mercury and Its Compounds
- Nickel and Its Compounds
- Odorous Compounds
- Organic Carcinogens
- Pesticides
- Phosphorus and Its Compounds
- Radioactive Substances
- Selenium and Its Compounds
- Vanadium and Its Compounds
- Zinc and Its Compounds

These reports represent current state-of-the-art literature reviews supplemented by discussions with selected knowledgeable individuals both within and outside the Federal Government. They do not however presume to be a synthesis of available information but rather a summary without an attempt to interpret or reconcile conflicting data. The reports are necessarily limited in their discussion of health effects for some pollutants to descriptions of occupational health exposures and animal laboratory studies since only a few epidemiologic studies were available.

Initially these reports were generally intended as internal documents within NAPCA to provide a basis for sound decision-making on program guidance for future research activities and to allow ranking of future activities relating to the development of criteria and control technology documents. However, it is apparent that these reports may also be of significant value to many others in air pollution control, such as State or local air pollution control officials, as a library of information on which to base informed decisions on pollutants to be controlled in their geographic areas. Additionally, these reports may stimulate scientific investigators to pursue research in needed areas. They also provide for the interested citizen readily available information about a given pollutant. Therefore, they are being given wide distribution with the assumption that they will be used with full knowledge of their value and limitations.

This series of reports was compiled and prepared by the Litton personnel listed below:

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ABSTRACT

Biological aerosols—suspensions of microorganisms in the air—can cause diseases in humans, animals, and plants and degradation of inanimate materials. Several typical airborne infections of humans include tuberculosis, pneumonia, the common cold, and influenza. In addition, there is evidence that biological aerosols and nonbiological air pollutants may act synergistically to produce harmful effects. Some airborne diseases of animals are tuberculosis, hog cholera, and Newcastle disease. Plants are susceptible to airborne pathogens that cause such diseases as wheat rust, potato blight, and almond brown rot. Organic constituents of protective paint coatings and other inanimate surfaces are subject to microbial attack and damage. The present knowledge pertaining to the relationships between dose-effect, viability, survival of microorganisms in aerosols, and other factors is insufficient for establishing standards for either indoor or outdoor environmental air concentrations.

The source of most human and animal airborne pathogens is the host organism that recently harbored the pathogens. However, since biological aerosols generally are detrimentally affected by exposure to the atmosphere, they are usually found in spaces close to the host. However, certain plant pathogens are more resistant to the atmospheric environment, and these are often rapidly dispersed hundreds of miles by air within a few days.

The abatement and control of biological aerosols have been successful only in environmentally-controlled indoor spaces. There has been no adequate way to estimate either the cost of the effects of biological aerosols, or the cost of abatement and control.

The available methods of analysis for biological aerosols tend to be specialized according to atmospheric conditions, biological types, and particle size; consequently, many different individual sampling devices are used.

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1. INTRODUCTION

Biological aerosols are defined as biological contaminants occurring as solid or liquid particles in the air. These particles can vary in size from virus units of less than 0.1 μ* to fungal spores of 100 μ or larger. The particles may occur as single, unattached organisms or as aggregates of organisms. They may also adhere to a dust particle or be surrounded by a film of dried organic or inorganic material. Viable microorganisms are known to occur up to an altitude of about 20 miles, and fungal spores have been found in air flights over the North Pole. For the purpose of this report, emphasis will be on those micoorganisms occurring as aerosols that can be pathogenic for humans, animals, and plants and can cause damage to inanimate materials. The microorganisms (microbes) generally involved are bacteria, fungi (yeasts and molds), and viruses. Other microorganisms—such as algae, protozoa, and rickettsiae—generally do not cause disease by transmission as aerosols. Some fungi have been implicated in hypersensitivity (allergic) reactions in humans, but this subject is discussed in a separate report of this series, "Air Pollution Aspects of Aeroallergens."

The importance of disease transmission by biological aerosols has been in part a function of urbanization. Because microorganisms do not generally survive very long as aerosols, airborne transmission of human and animal diseases is limited to indoor or crowded outdoor spaces. The appearance of crowded cities in what had been primarily a low population-density agricultural society for the previous 1,000 years was a contributing factor in the plague epidemic of 1348. Since then, respiratory diseases have been correlated with the extent of crowded conditions in the cities. Although progress in modern medicine dramatically decreased the potential mortality rate during the influenza pandemic of 1968, the incidence of the disease demonstrated that we are far from an adequate control of such diseases.

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*μ=micron(s).

2. EFFECTS

Biological aerosols have been shown to produce diseases in humans, animals, and plants and microbial degradation of inanimate materials. When airborne dissemination is involved in transmission of diseases, the actual number of microorganisms dispersed by the host is relatively small, and if dispersed into the open air, the living organisms represent a very small fraction of the total ambient air. These pathogenic microorganisms cannot reproduce in the air and generally do not survive long because of adverse conditions of humidity, temperature, and sunlight. Airborne transmission of human and animal pathogens is therefore essentially limited to indoor spaces or to closely confined outdoor spaces. The general exceptions to this fact are certain microorganisms that multiply saprophytically in the soil and can be pathogenic to humans and animals. These microorganisms are not dependent upon a host reservoir for survival and can be more widely dispersed by air. In general, the symptoms produced by airborne infectious agents are those of a respiratory disease, but transmission often can be by other means and results in symptoms other than respiratory ones.

2.1 Effects on Humans

2.1.1 Airborne Diseases

2.1.1.1 Bacterial Diseases

The most common airborne bacterial infections of humans are the following:[12,17,38,95,105,112,114]

- Pulmonary tuberculosis
- Pulmonary anthrax
- Staphylococcal respiratory infection
- Streptococcal respiratory infection
- Meningococcal infection
- Pneumococcal pneumonia
- Pneumonic plague
- Whooping cough
- Diphtheria
- Klebsiella respiratory infection
- Staphylococcal wound infection

The causative agent and the symptoms of each of these diseases are presented in Table 7 in the Appendix.

2.1.1.2 Fungal Diseases

The most common airborne fungal infections of humans are the following:[12,17,38,95,105,112,114]

- Aspergillosis
- Blastomycosis
- Coccidioidomycosis
- Cryptococcosis
- Histoplasmosis
- Nocardiosis

The causative agent and symptoms of each of these diseases are presented in Table 8 in the Appendix.

2.1.1.3 Viral Diseases

The most common airborne viral respiratory diseases of humans are these:[12,17,38,51,91,95,105,112,114]

- Influenza
- Febrile pharyngitis or tonsillitis
- Common cold
- Croup
- Bronchitis
- Bronchiolitis
- Pneumonia
- Febrile sore throat
- Pleurodynia
- Psittacosis

More than 90 viral agents have been identified as the etiological factor in respiratory tract illnesses. Others remain yet unrecognized by present laboratory techniques, which at best identify the causative viral agent in only 50 to 60 percent of respiratory infections. Table 9 (Appendix) lists the names of the viral, the one rickettsial (Q-fever),[64,113] and related diseases in which airborne transmission is primarily involved.

In addition to those diseases listed in Table 9, several viral diseases in which airborne transmission is at least partly involved produce symptoms other than respiratory ones. These are as follows:[17,91,105,112]

Mumps is a swelling and tenderness of the parotid glands that is sometimes accompanied by orchitis.

Rubella (German measles) is a mild exanthematous disease of childhood resembling measles. However, its occurrence in women during the early months of pregnancy is associated with a high incidence of congenital malformations.

Rubeola (measles) is the commonest disease of childhood. The median number of cases reported per year in this country exceeds half a million. Rubeola is characterized by a cough and fever and a macular or maculopapular rash tending to become confluent.

The transmission of several other viral diseases can be due in part to airborne contaminated dust and skin scales:[12,17,91,105]

Variola (smallpox) is characterized by vesicles over most of the skin surfaces. Although it is endemic elsewhere in the world, no confirmed cases have been reported in this country since 1954.

Varicella (chickenpox) is a common, highly contagious, exanthematous disease of childhood that occurs in epidemic form.

Herpes zoster (shingles) is similar to varicella but occurs more frequently in adults.

2.1.1.4 Hypersensitivity Reactions

A number of fungi, and possibly algae as well, have been implicated in hypersensitivity (allergic) reactions in humans. Evidence has been presented in recent years that the symptoms of such diseases as farmer's lung, mushroom lung, and other diseases formerly considered to be infectious were the results of hypersensitivity responses. These syndromes will not be discussed in this report but are included in another report in this series, "Air Pollution Aspects of Aeroallergens."

2.1.2 Synergistic Effects

As modern air pollution information has accumulated, it has become apparent that increases in respiratory infection morbidity and mortality of the exposed population may be related to excessively high levels of nonbiological air pollutants. That is, the two types of pollutants—biological and nonbiological—may produce synergistic or potentiating effects.

However, as so often in the study of natural human infections, one is limited to conclusions based upon medical statistics on uncontrolled events and situations. The number of model situations in which high levels of air pollution were accompanied by marked increases in respiratory disease morbidity and mortality and adverse weather conditions has been limited. Conditions in the laboratory with experimental animals are subject to better control, but there are, nevertheless, limitations in applying results to human disease processes.

Several reports are available on the potentiation of air pollution and influenza. Both high levels of air pollution and influenza occurred in London in December 1952 in which 4,000 deaths occurred,[73,87] and again in 1958 to 1959.[65] The observations indicated a parallel correlation between the increase in air pollution and an increase in the disease. Greenburg et al.,[44] in a study of pediatric and adult clinic visits, found an increase in upper respiratory illness during the New York City air pollution incident of November 1953. They investigated the influenza epidemic in New York City during the fall of 1957 but could not ascertain quantitative relationships between air pollution and influenza.[45] However, during the period of January 29 to February 12, 1963, another occurrence of influenza in New York City did show a correlation with air pollution. During this period, 809 deaths occurred in excess of the overall average number of deaths for the same 15-day periods in 1961, 1962, 1964, and 1965. This increase in mortality took place primarily in the older age groups—45 to 64, and 65 and over.[45] Dohan[25] reported a decrease in respiratory illnesses in Pittsburgh in the years following the intensive efforts to control air pollution.

Douglas and Waller[26] in 1966 reported on a study of a group of schoolchildren performed as part of the National Survey of Health and Development in Great Britain. The purpose of the study was to examine the relationship between respiratory infections and prolonged exposure in areas of high and low air pollution. Douglas and Waller followed the medical histories of 5,362 children born during the first week of March 1946 until they reached the age of 15 in 1961. At that time 4,592 were still living in Great Britain, and complete medical records were available for 3,866. The investigators concluded that upper respiratory tract infections were not related to the amount of air pollution, but that lower respiratory tract infections were. Also, the frequency and severity of the lower respiratory tract infections increased with the amount of air pollution exposure, and both boys and girls were affected equally. An association between lower respiratory tract infection and air pollution was found at each age examined (6,7,11, and 15 years). There were no differences observed between children in middle- and working-class families.

Lunn *et al.*[63] collected respiratory illness data on 819 schoolchildren between 5 and 6 years old who had lived in the Sheffield area most of their lives. These investigators reported a relationship between both upper and lower respiratory illnesses and air pollution. However, socioeconomic factors—such as social class, number of children in the house, and and sharing of bedrooms—appeared to have little influence upon the respiratory illnesses among the children.

Alderson[2] presented data from the British Ministry of Pensions and National Insurance which showed that different illness patterns existed among individuals of three occupations. Coal miners had more respiratory illnesses than professional and technical personnel, who in turn had more than agricultural workers. The illness patterns are presented in part in Table 1.

The results of animal experiments in relation to synergistic effects are discussed in Section 2.2.2, Experimental Animals.

TABLE 1

AGE-STANDARDIZED INCEPTION RATES OF INCAPACITY AMONG MEN IN THREE DIFFERENT OCCUPATIONAL GROUPS[2]

	Men Incapacitated per 100 at Risk		
Diagnosis	Agricultural Workers	Coal Miners, Face Workers	Professional and Technical Personnel
Acute upper respiratory infection	40	284	87
Influenza	60	234	80
Bronchitis	47	205	51

2.2 Effects on Animals

2.2.1 Commercial and Domestic Animals

Few diseases of commercial and domestic animals can be attributed to airborne aerosols. Most animal diseases are transmitted by contact by insect bites, and through ingestion of contaminated food and water.

2.2.1.1 Bacterial Diseases

Two bacterial airborne diseases of animals are tuberculosis (Mycobacterium bovis) of cattle, swine, sheep, dogs, and cats, the control of which in the United States has been by slaughter; and glanders (Actinobacillus (Malleomyces) mallei), a tuberculosis-like, high-mortality disease of horses, mules, and asses.[11,69,70]

2.2.1.2 Fungal Diseases

Several fungal diseases of animals that may possibly be airborne are the following:[11,69,70]

Aspergillosis occurs in domesticated birds, pigeons, ducks, and chickens. It can occur as a superficial infection of the air sacs, which become covered with a mat of green mycelium; a nodular tubercle-like mass; or a diffusely infiltrated pneumonic infection of the lung. In chicks, an epidemic form is known as "brooder pneumonia," the source being inhalation of grain and straw heavily contaminated with mold. A nodular or pneumonic form can occur also in cattle and sheep, and especially in horses.

Cryptococcosis can occur in the lungs of horses and result in granulomas. Emmons[35] found virulent strains of Cryptococcus neoformans in pigeon manure. It has also been described as the etiological agent in a severe outbreak of bovine mastitis.[12]

Coccidioidomycosis is endemic in areas of the Southwest United States. It occurs naturally in domestic animals, including cattle, horses, sheep, swine, and dogs, and also in certain wild rodents (pocket mouse, kangaroo rat, and grasshopper mouse).

2.2.1.3 Viral Diseases

The most common viral diseases of animals are as follows:[11,69,70]

Hog cholera
Equine influenza
Swine influenza
Feline distemper
Canine distemper
Newcastle disease
Infectious bronchitis

The symptoms of these diseases are presented in Table 10 in the Appendix.

2.2.2 Experimental Animals

2.2.2.1 General Experiments

Table 11 (Appendix) lists the airborne bacterial and fungal diseases and the common laboratory animals which have been used in aerosol studies. However, the use of laboratory animals—mice, rabbits, guinea pigs, and monkeys—has generally been limited in virus aerosol studies because of the specific host-parasite relationship of viruses.

2.2.2.2 Synergistic Experiments

Because of the difficulties in studying the potential synergistic effects of nonbiological and biological air pollutants on humans, many experiments have been made using experimental animals exposed to mixtures of artificially produced aerosols under the relatively controlled conditions of the laboratory.

Miller and Ehrlich[71] studied the effect of ozone on susceptibility to respiratory infection in mice exposed to aerosols of Klebsiella pneumoniae and various streptococcus species. The mice were also exposed to ozone concentrations of 0.4 to 4.4 ppm for periods ranging from 3 to 100 hours. The time between ozone exposure and subsequent aerosol exposure or challenge was 1 hour. Exposure to ozone significantly reduced resistance to infection as measured by mortality rate and survival time. In a later study,[88] it was found that the mice's resistance was reduced for as long as 19 hours between ozone exposure and aerosol challenge. Coffin and Blommer[15] reported that exposure to 0.7 to 0.9 ppm ozone for 2 hours enhanced mortality to streptococcal pneumonia in mice. The exposure to the streptococcal aerosol occurred 30 minutes following the ozone. The mortality rate was further enhanced by exposure of the mice to a cold temperature (6 to 9° C) for 3 hours prior to the ozone and streptococcus aerosol. The authors believed that the effect of the cold temperature was to potentiate the ozone effect. Thienes _et al_.[110] were unable to demonstrate a potentiation effect between ozone and tuberculosis in mice.

Ehrlich[32] has reviewed the effects of nitrogen dioxide (NO_2) on the resistance of laboratory animals to _K_. _pneumoniae_ infections. A single 2-hour exposure of mice to 3.5 ppm of nitrogen dioxide before or after respiratory challenge with aerosol of _K_. _pneumoniae_ significantly increased mortality. To produce the same effects in hamsters and squirrel monkeys, 35 ppm was required. The effect of the single 2-hour exposure was not persistent, and it was observed that normal resistance to the infection returned within 24 hours after cessation of the exposure to nitrogen dioxide. Continuous exposures to 0.5 ppm for 3 months or longer, as well as intermittent daily exposures over a 30-day period, produced the same effect in mice as the single 2-hour exposure to 3.5 ppm. Intermittent exposure of mice to 0.5 ppm for 6 to 18 hours per day for 6 months also resulted in a significantly increased mortality.[33]

Henry _et al._[49] in studying the combined effects of nitrogen dioxide and _K_. _pneumoniae_ microorganisms on squirrel monkeys, reported that a combined stress of 50 ppm NO_2 and 10^4 cells of _K_. _pneumoniae_—neither in itself fatal—produced death. At NO_2 concentrations of 35 ppm or less, death did not occur, but bacterial clearance from the lungs was delayed or prevented. Monkeys exposed only to the challenge dosage of _K_. _pneumoniae_ showed no bacteria in their lungs 15 to 57 days following challenge. However, if preceded with 10 ppm NO_2, _K_. _pneumoniae_ could be found in the lungs 19 to 51 days later.

Coffin and Blommer[16] have reported results indicating that light-irradiated automobile engine exhaust enhanced the pneumonia mortality rate of mice exposed to a streptococcal aerosol.

Inert dust particles have been reported to potentiate infections in laboratory animals. Tacquet[109] observed an increase in pathogenicity and in the number of mycobacteria isolated from the lungs of guinea pigs following inhalation of inert carbon dust. Laurenzi[61] reported that the natural clearance of aerosolized staphylococci from the lungs of mice was impaired by inhalation of cigarette smoke or intraperitoneal injections of ethyl alcohol. Green and Kass[42] have made similar observations.

2.3 Effects on Plants

Plants are susceptible to bacterial, fungal, and viral diseases. Some of these diseases are disseminated by means of insects, birds, animals, or water; but many—primarily the fungi—are subject to airborne dispersal. The following is a list of such airborne plant diseases:[3,14,22,90,124,129]

- Almond brown rot
- Azalea flower spot
- Beet downy mildew
- Blossom infection
- Cedar rust
- Apple rust
- Chestnut blight
- Crown rust of oats
- Downy mildew
- Leaf spots on tulips
- Loose smut of wheat
- Maize rust
- Onion mildew
- Potato late blight
- Powdery mildew on barley
- Stem rust of wheat and rye
- Tobacco blue mold
- White pine blister rust

2.4 Effects on Materials

Microorganisms are essential in normal decay processes. Therefore, all material surfaces in contact with the air are theoretically subject in some degree to microbial degradation by saprophytic microorganisms. The most obvious general example of this is food spoilage, a continual problem. The magnitude of this problem is related to the local climate: maximum spoilage occurs in a hot, humid climate and minimum spoilage in a cold, dry climate.

Saprophytic fungi can grow on the surfaces of many inanimate materials where there is high humidity. The organisms may utilize either the coating or the underlying surface as food, and may produce corrosive acid or alkali wastes as a result of their metabolic processes. These wastes may in turn attack the surfaces on which they are growing. For example, the modern field of miniaturized electronics is faced with this problem: the miniaturized circuits, unless protected by varnishes containing fungicides, can be damaged by the growth of fungi.[29] Larsen[60] in 1957 pointed out that organic constituents of protective paint coatings may be subject to microbial attack and damage.

2.5 Environmental Air Standards

There are no environmental air standards applicable to biological aerosols at the present time. Current knowledge pertaining to the relationship between dose-effect, viability, survival of microorganisms in aerosols, sampling procedures, and aerosol production is insufficient for establishing standards for either indoor or outdoor environmental air concentrations.

3. SOURCES

3.1 Natural Occurrence

Microorganisms are ubiquitous in nature. However, all microorganisms found in the air had as their original habitat either soil, water, humans, animals, or plants. Microorganisms become airborne from the soil and from plants by wind disturbances, and from water by wave and wind action. They come from animals through shedding, excreta, and respiratory droplets, and from humans through shedding from skin and clothing and through respiratory droplets produced by speech, coughing, and sneezing. Some types of organisms are more plentiful in the air than others, because of their size (i.e., they are small enough to remain airborne), the magnitude of the emission source, the death rate of organisms suspended in the air, and other factors. Microorganisms have been found at various altitudes.[50] Fulton[39] in sampling the air above San Antonio, reported average peak concentrations of 250, 75, and 35 microorganisms per cubic meter at 690, 1,600, and 3,127 meters' altitude respectively (Table 12, Appendix). Microorganisms have also been recovered in balloon flights up to 90,000 ft.[10] (Table 13, Appendix).

Most of the microorganisms found in the air are saprophytic and generally are not pathogenic. Those which are pathogenic, with some exceptions, come from a living host. Since they are usually detrimentally affected by exposure to the atmosphere, they are only found in close proximity to the host. However, certain plant pathogens can have rapid and widespread aerial dispersal over hundreds of miles within a few days.[107]

The survival of biological aerosols has been studied rather intensively in recent years, primarily in laboratory studies. Most of these studies have been concerned with bacteria, and relatively little is known of the behavior of viruses and fungi. No simple relationship has been found between the degree of survival and age of the aerosol. The half-life of the aerosol is affected by such variable factors as the species of microorganisms (spore-former or non-spore-former); metabolic state of the microorganism; the relative humidity, gaseous composition, and temperature of the air; radiation; collection method; and others. Because of the large number of these variables and their interrelationship, both the results and the interpretations of aerosol survival studies are markedly dependent upon the precise technique employed.[5]

3.2 Production Sources

The spread of influenza, the common cold, and other such diseases in the home, office, or schoolroom is readily apparent. Similar disease transmission takes place in hospitals as well. For example, the spread of staphylococcal infections in hospitals has been a considerable problem. Staphylococcus aureus is commonly found in the normal nasal flora of 30 to 50 percent of healthy adults (carriers).[126] Studies on nasal carriers showed that while direct dispersal did not take place to any great extent under normal conditions, large numbers of infectious airborne particles might be produced by active movements.[104] The bedclothing of carriers also becomes rapidly infected.[77,96] Wilkoff et al.[125] studied the viability of Staphylococcus aureus dispersed by aerosol on various fabrics (wool blanket, wool abardine, cotton sheeting, cotton knit jersey, cotton terry cloth, and cotton wash-and-wear material). He found that staphylococcal populations persisted long enough (4 to 24 weeks) to be of epidemiological importance. Davies and Noble[21] observed under a microscope that airborne particles from a hospital ward included many skin scales containing staphylococci. In addition, they found that the skin scales and bacterial content of the air rose significantly during bedmaking.

Eichenwald et al.[34] have described a direct dispersal of smaller than normal ($< 5\ \mu$) particles containing staphylococci from the upper respiratory tract of newborn infants in a nursery. These "cloud babies" had a respiratory virus infection and, apparently because of the slightly restricted air passages, were producing "clouds" of staphylococci. Staphylococci are commonly found on the healthy skin and, therefore, skin desquamation is an important source of hospital staphylococci. Some individuals are prolific dispersers.

Airborne dust containing streptococci has been found in hospital wards containing patients with streptococcal infections. M. tuberculosis has also been found in the dust of sanitariums, and the diphtheria organism in floor dust near diphtheria patients.[114]

Gip[40] was able to isolate airborne dermatophytes from a commercial bathhouse, a dressing room in an automobile factory, and a hospital, as well as in a gymnasium during a basketball game. However, the role of such isolated dermatophytes as exogenous agents of fungus infection is still open to question.

Procknow[86] in 1967 reported isolating Histoplasma capsulatum annually for 15 years from the dust of an unused silo. At the time of its abandonment in 1950, the silo was the source of histoplasmosis contracted by a farm family of six.

Emmons[36] was able to isolate H. capsulatum from all 10 soil samples collected in a downtown park in Washington, D.C. He attributed the presence of the fungus to droppings from starlings roosting in trees.

D'Alessio[20] reported an urban epidemic of histoplasmosis which occurred in Mason City, Iowa, in 1962. The source of the fungus, proved by the recovery of the organism from the soil, was a starling roost in the center of town. The airborne epidemic had occurred after bulldozing of vegetation in the area had produced clouds of dust. It was concluded that about

2,400 schoolchildren and about 6,000 adults had been infected during the epidemic.

Virulent strains of Cryptococcus neoformans have been found in pigeon manure in old pigeon nests and under roosting sites.[35] The organism was isolated from 63 of 91 specimens obtained in and around Washington, D.C. (Table 2).

TABLE 2

ISOLATION OF CRYPTOCOCCUS NEOFORMANS[35]

Sources of Collection	Number of Specimens Collected	Number of Specimens Positive
Warehouse, former barn	15	14
Old school building, now offices	10	7
Grain mill establishment	5	3
Cupola on high school building	7	7
Window ledges, Federal and municipal office buildings	18	17
Public parks	7	0
Railroad station	4	1
Barns (Virginia and Maryland)	25	14
Total	91	63

Wells,[121] in the 1930's, concluded from his studies that bacterial contamination of air by sewage works existed and that organisms causing respiratory diseases could remain airborne and viable for long periods of time. Randall and Ledbetter[89] sampled the air of an activated sludge sewage treatment unit and found that 6 percent of all bacteria emitted by the waste liquid were of the Klebsiella species, potential respiratory tract pathogens. About 40 percent of the viable airborne bacteria in the immediate vicinity of the activated sludge units were of a size that permits lung penetration (5 μ or less). The bacterial population persisted for a considerable time and distance (the farthest sampling point being at 100 feet); the distance was strongly dependent on the wind velocity.

Napolitano and Rowe[76] sampled the air of sewage treatment plants and found that in one plant, the unit discharging most organisms was the aeration tank. In a second plant, the comparable units were the trickling filters. Emitted bacteria were found at the farthest sampling point, 150 feet downwind of the unit. The investigators did not attempt to isolate pathogens per se.

Albrecht[1] demonstrated that the distance traveled and the number of bacteria found downwind of a trickling filter were correlated directly with the wind velocity. Jensen[56] surmised from his studies that tuberculosis organisms could become airborne from liquids in a sewage plant and were a real danger to the operating and supervisory personnel of the plant. Dixon and McCabe[24] attempted to determine whether the incidence of infection in sewage plant workers had increased, but the results were inconclusive because of incomplete employee medical records.

Spendlove[106a] has studied the aerosol production in an animal rendering plant. He painted slurries of harmless tracer bacteria (a spore-former and a non-spore-former) onto the carcasses before the rendering process began, and later collected air samples at various places inside and outside the plant as processing proceeded. His results showed that the rendering process in use created aerosols of viable microorganisms. Both the vegetative and spore-forming tracer organisms were found in air samples taken inside the plant and at 100 feet downwind from the exhaust stack. These findings supported the suspicion that some of the workers in the plant had become infected with ornithosis at an earlier date when diseased turkeys had been processed. Other diseases which potentially could have been transmitted by this rendering plant include anthrax, brucellosis, tularemia, glanders, sylvatic plaque, Q fever, and virus equine encephalitis. This situation was a health hazard both to the workers within the plant and to the population in surrounding areas.

Many microorganisms—bacteria, yeasts, and molds—are used in industrial fermentations to produce a number of economically important materials. The latter include butanol, acetone, ethanol, vitamins B_2 and B12,lactic acid, amylase, dextran, diacetyl, acetic acid (vinegar), antibiotics, industrial alcohols, beverage alcohols, citric acid, corticosterone, and gibberellin, as well as dairy products such as butter, cheese, and various fermented milks.[13,38] However, even though huge quantities of microorganisms are involved in the production of these materials, no information was found on these fermentations as a source of outdoor or indoor air pollution. Ashe[6] has stated that to his knowledge, no industry has been reported to produce a disease in the general population through air pollution by living organisms.

Production of vast numbers of spores in periodic waves is a characteristic of many fungi, and the retention of viability is of fundamental importance, especially during prolonged air transport. High temperature, radiation, and low humidities may have an adverse effect on spores of many of the airborne fungi. Full sunlight is known to decrease the viability of many plant pathogens.[126] Failure to demonstrate high germination rate may not be from lack of viability, but from a lack of nutrient,[52,81] or presence of inhibitors[129] and factors still unknown.

Murrow _et al_.[74] have summarized the most frequently isolated molds from 41 sampling stations across the country. No two stations had the same lists, but a basic group of dominant genera appeared to occur. These were the following:

Alternaria
Homodendrum
Aspergillus
Penicillium
Pullularia
Phoma
Trichoderma
Fusarium
Helminthosporium
Cryptococcus
Rhodotorula

Similar genera of fungi were observed in Tucson and Phoenix, Ariz.,[31,41] in Albuquerque, N.Mex.[30] and in Los Angeles, Calif.[103]

Altman *et al.*[3] have tabulated the observations of many investigators on dispersal parameters of fungi pathogenic for plants. Their tabulation is reproduced in part in Table 14 (Appendix).

The extent to which pathogenic fungi will spread is dependent upon the occurrence of those particular conditions—of humidity, temperature, winds, and presence of plant host—that favor a particular disease. A classical example has been described by Stakman and Harrar[107] in which all conditions were favorable for wheat rust disease. In 1935, spring was late, and in northern Texas the rainfall was twice the normal amount. Rust (*Puccinia graminis tritici*) developed quickly. Spores of the fungus were blown northward and encountered favorable conditions for development in the late crops of Kansas and Nebraska. Furthermore, cold weather in May and June had delayed the wheat crop in Minnesota and North and South Dakota. The first half of July was still wet, but hot, and when the masses os spores were blown into these fields from Kansas and Nebraska, wheat rust developed in epidemic proportions. It is estimated that 135 million bushels of wheat were lost in Minnesota, North Dakota, and South Dakota alone.

Species of algae and protozoa have been reported as making up part of the aerial biota.[67,98,99] Viable samples have been obtained under extreme environmental conditions, including rain, heavy snow, and fog during fall, winter, and spring. Brown _et al._[9] found that the quantity of algal cells in the air exceeded that of mold spores. However, algal cells have not been known to cause any infectious disease, and their role as aeroallergens has yet to be definitely established.

3.3 Product Sources

Although large quantities of microorganisms are produced as a result of various industrial fermentations, Ashe[6] stated in 1959 that there has been no evidence so far that this has resulted in a health hazard to the general population. Except for the observations of Spendlove[106a] (see Section 3.2), no other information relating to this point has been found in the literature.

3.4 Environmental Air Concentrations

It is not valid to present any one set of values for the aerial microbial concentration of a given area, such as a schoolroom or a playground. Any count is influenced by the temperature, meteorological conditions, vegetation, human and animal population, and time of day, as well as by the inability to determine all types of microorganisms by any one sampling procedure. With due consideration to the latter fact, the following are some values which have been reported. Table 3 presents the bacterial counts of several areas obtained in New York City in 1936.[127] Figure 1 (Appendix) presents mean counts of outside air obtained in Detroit for a 3-month period in 1953.[127] Air samples collected during the winter indicated a lower concentration of airborne bacterial particulates than in the spring. Hourly fluctuation in counts for air samples collected in an open field of a nonurban area in Georgia in 1951 is shown in Figure 2 (Appendix). The fluctuations in the number of airborne microorganisms in a surgery room due to movement are presented in Figure 3 (Appendix).

Wright _et al_.[127a] have reported the results of a pilot study to evaluate the types and number of viable microorganisms present in the air of an urban area such as Minneapolis-St. Paul. Air samples were obtained at four points (35, 70, 170, and 500 feet) along a 500-foot television tower by means of an Anderson sampler. Sampling was performed at intervals over a 6-month period, and wind, rainfall, humidity, and temperature conditions were recorded with each sample. The mean viable counts were as follows:

58 particles per ft^3 (2,047 per m^3) at 35 ft
38.4 particles per ft^3 (1,355 per m^3) at 75 ft
32.7 particles per ft^3 (1,155 per m^3) at 170 ft
22.4 particles per ft^3 (790 per m^3) at 500 ft

The range of all counts observed was 3.5 particles per ft^3 (123 per m^3) to 141 particles per ft^3 (4,977 per m^3), with no consistent relationships between the counts and any of the meteorological parameters. Regardless of altitude, molds constitued approximately 70 percent of the total airborne microflora, bacteria between 19 and 26 percent, and yeast and actinomycetes the remainder. A significant portion of the viable microorganisms in the air were in the particle size range of 3 to 5 μ.

Microbial counts in nonurban areas are usually relatively lower than in urban areas, and in both areas are influenced primarily by the degree of activity and dust in the immediate area as well as by seasonal and climatic conditions.

TABLE 3

BACTERIA IN AIR IN NEW YORK CITY, JANUARY-JUNE 1936[127]

Location	No. of Samples	No. of Bacteria per ft^3	No. of Streptococci per ft^3		
			All Types	Beta Hemolytic	Alpha Hemolytic
Indoor					
Schools	707	29.6	0.20	0.01	0.18
Subway	290	19.2	0.10	0.0003	0.085
Theater (nonventilated)	104	13.2	0.04	0.001	0.38
Theater (ventilated)	149	3.1	0.03	0.0005	0.26
Outdoor					
Streets	143	11.2	0.05	0.0001	0.45
Park	13	3.0			

Randall and Ledbetter,[89] in sampling the air of an activated sludge sewage treatment unit, found an increase from about eight viable particles per cubic foot (283 particles per cubic meter) on the upwind side to 1,170 per cubic foot (17,900 per cubic meter) on the downwind side. Figure 4 (Appendix) shows the decrease in numbers with distance downwind from a treatment unit.[62]

The number of airborne fungi changes from season to to season, from day to day, and even from hour to hour. Table 4 illustrates the average hourly fluctuation observed by Pathak and Pady[84] of Alternaria spores sampled in Manhattan, Kansas. Some fungi appear to have a diurnal periodicity.[83,84] One explanation offered for the latter fact is that a single crop of spores—of Cladosporium, for example—is produced per 24-hour period, maturing at night and ready to be released just before daylight. Morning turbulence carrying the spores into the air for a monitoring peak, e.g., 100 per cubic foot (3,500 per cubic meter). Decreasing air turbulence later in the day allows the spores to settle, producing a late afternoon or early evening peak.[92]

TABLE 4

AVERAGE NUMBER OF ALTERNARIA SPORES AT ONE SITE IN MANHATTAN, KANSAS[84]

Time	Number per Ft^3
5 a.m.	12
6	7
7	9
9	13
11	16
1 p.m.	17
2	13
5	16
6	19

Pady[82] found fungus spores present in the atmosphere at an elevation of 150 feet throughout the year at one site atop a building in Manhattan, Kans., with peaks in July and August. In summer the number varied from 50 to 700 particles per cubic foot (1,765 to 24,700 per cubic meter), while in winter they ranged from 5 to 20 per cubic foot (175 to 700 per cubic meter). *Cladosporium* was present throughout the year, comprising the bulk of the spores in summer.

4. ABATEMENT

The problem of abatement and control of biological aerosols and their effects is a most difficult one. In general, knowledge is incomplete concerning the various parameters of biological aerosol production, the survivial and transmission of the aerosol, the sampling procedures, and other factors. There are further complications when air is not the only route by which a given pathogen is spread. It is often difficult to decide just how and when some infections were acquired. In addition, the quantitative nature of the dose-effect relationship is influenced by both the host and the pathogen, as well as by the possible synergistic relationships with other pollutants.

The abatement of some diseases, such as influenza, is of such complexity that some researchers believe that control will be dependent upon individual protection by immunization.[108] However, attempts have been made to control airborne infections indoors by the use of ultraviolet light. Wells et al.[123] reported success in the control of a measles epidemic in Philadelphia in 1941 by irradiating the air of classrooms with ultraviolet light. Perkins et al.,[85] however, found several years later that similar attempts at irradiation of classroom air did not reduce the incidence of measles. The early success of Wells was attributed to the social structure of the communities where ultraviolet light was used; apparently the transmission of measles took place primarily at school. In later studies, Wells and Holla[122] and Wells[121] attempted to approach the broad problem of airborne infections on a community-wide basis. They attempted to irradiate with ultraviolet light the air of public buildings—schools, churches, a theater, clubs, certain stores, and other places where children gathered—in Pleasantville, N.Y. A neighboring community served as a control. The results after 4 years showed that the irradiation had little effect upon the total incidence of airborne infections. In another study, however, ultraviolet light was used successfully to control influenza in a hospital building.[95] One building was irradiated while a similar building was not. No attempt was made to control the hospital staff working in the two buildings. After 8½ months in 1957 to 1958, 2 percent of the 209 patients in the irradiated group had contracted influenza as compared to 19 percent of the 396 patients in the control group. Ultraviolet irradiation has also been used successfully in special situations, such as above a surgery table.

The control of hospital-acquired infections, especially staphylococcal infections, has become a problem of considerable magnitude. There is evidence that suggests that the inhalation of airborne bacteria in dust has a greater quantitative effect than inhalation of directly expelled particles in producing disease.[12] Therefore, control measures directed toward the suppression of dust have been employed, such as use of particle-retaining oils on blankets and floors in hospitals. In addition, the use of residual disinfectants, more frequent changes of bed coverings, and the use of different fabrics have helped control transmission.

Selwyn[102] found that for those spreading staphylococcal organisms, treatment of the skin with antibiotics greatly reduced both dispersal of staphylococci and the risks of acquisition of the organisms by new patients. Solberg[106] found the same to be true for nasal carriers in hospital wards. Washing the skin with hexachlorophene-containing soaps also reduced skin dispersal of staphylococci.[106]

The use of disinfectants to control undesirable microorganisms in hospitals and elsewhere is common. However, the disinfectants must be correctly used. Table 15 in the Appendix, from Jemski and Phillips,[54] lists some common germicides and conditions for their use.

High-speed photography has dramatically demonstrated the value of surgical masks in reducing the number of particles emitted during a sneeze.[55] However, to minimize discomfort in wearing them and to improve retention efficiency, newer masks are being developed and tested. Guyton and Deker[47] tested the efficiency of masks of different designs. One type designed for resterilization and reuse had a filtering efficiency for airborne particles (1 to 5 μ diameter) of 99 percent. Two of the disposable types had an efficiency of greater than 80 percent.

Healthy hospital personnel have been shown to be carriers and dispersers of staphylococci. Control of this problem has been accomplished either by antibiotic therapy, use of masks, or removal of these personnel from their positions in the hospital.

Within recent years, a number of air-filtration devices have become commercially available that are capable of removing extremely small particles, including microorganisms. These devices have been produced in different sizes and efficiencies. Units as small as face masks and helmets and others large enough to be used in air-conditioning systems are available, with efficiencies of up to 99.999 percent for removal of submicron particles.[23,48,119] These filters have been used to remove microorganisms from air in hospitals, commercial fermentation plants, and other controlled environmental systems.

In designing a filter system for a controlled environment, the relative position of the blower and filter in the system is important to avoid leakage of unfiltered air. Figure 5 (Appendix) shows the positioning of the blower and filter both when the contamination is inside the room and when the contamination is outside the room.[23] To be of value in a controlled environment, a filter sytem need not be 100 percent efficient. Table 5 derived from a mathematical model

(Table 16, Appendix), presents microbial air concentrations in a 500-cubic-foot room using filters of different efficiencies and with different microbial loadings.[23] The tabulation indicates that good reductions in microbial numbers can be obtained even with less than 100-percent-effective filters, especially since roughing filters generally are used in conjunction with the higher efficiency filters. Some of the commercially available filters and their characteristics—efficiency, composition, etc.—are listed in Tables 17, 18 19, 20, and 21 in the Appendix. The ultra high-efficiency units are capable of removing 0.1 u viral particles.[48,119] The results of one series of tests are presented in Table 21 (Appendix).

Public health authorities have made recommendations for the control of some diseases for which the infectious agent can survive for extended periods of time in soil and dust. For example, in endemic areas of coccidioidomycosis or histoplasmosis, dust control measures—oiling of roads and planting of grass—should be practiced, or local areas should be sprayed with disinfectants. Individuals from nonendemic areas should not be brought into endemic coccidioidomycosis areas for work in dusty occupations, such as cotton picking or road construction. Control of pigeons and starlings should be attempted in areas where histoplasmosis or cryptococcosis are potential hazards. Protective masks should be worn

TABLE 5

ROOM CONTAMINATION IN ORGANISMS PER CUBIC FOOT AT END OF ONE HOUR AND AT STEADY STATE[23]

% Filter Efficiency[a]	Organisms being generated per minute[b]		
	1,000	10,000	100,000
30	3.80085 (4.00000)	38.00852 (40.00000)	380.08520 (400.00000)
60	1.99504 (2.00000)	19.95042 (20.00000)	199.50420 (200.00000)
90	1.33316 (1.33333)	13.33163 (13.33333)	133.31630 (133.33333)
100	1.19994 (1.20000)	11.99946 (12.00000)	119.99460 (120.00000)

[a] Assumptions: 5,000 cubic feet in room; clean at start. Then air changes 10 times per hour through filters. Complete mixing obtained at all times.

[b] First figure in the body of the table gives concentration in organisms per cubic foot reached at end of one hour. The second figure, in parentheses, gives the equilibrium or steady-state concentration. For development of the mathematical solution of this problem, see Table 16, Appendix.

by persons exposed to known or potential sources of infection, such as the cleaning or destruction of old buildings—chicken houses, barns, and silos, for example—where starlings and pigeons have roosted. All articles contaminated by persons or animals infected with blastomycosis, tuberculosis, and other such infectious diseases, as well as their sputum, should be disinfected prior to disposal.[53]

Ledbetter[62] has suggested that elimination of any potential biological aerosol hazards associated with sewage treatment units could be effected by enclosing the process and venting the waste air through an incinerator with the proper controls for trapping particles and gases. The currently available devices for the control of industrial emmisions are discussed in detail in the National Air Pollution Control Administration report[18] "Control Techniques for Particulate Air Pollutants."

The control of mildew and other fungi on painted surfaces has not been very successful.[100] Paint formulas with zinc, titanium, and tin have been able to retard somewhat the growth of fungi but have not been completely inhibitory.

The problem of food storage in recent years has been solved successfully by the use of refrigeration. Sulfur dioxide, benzoates, and other preservatives have also been beneficially employed.

There are incidents in which abatement procedures have been employed before the need was evident. That is, no information was available beforehand as to the extent, if any, of a problem, but abatement was attempted because of "common sense." For example, in one report, the requirement of counterguards for protecting food from aerosols in cafeterias seems to have arisen without any specific data to show the need for it. A study[68] was performed to determine whether the general existing guard designs were of any value. The data did indicate that guards were of value in shielding food from potential aerosols being dispersed by the patrons. However, it is still not known how extensive this problem can be and whether the presently used designs give sufficient protection.

Research is continuing to develop fungus-resistant varieties of crops. For example, a rust-resistant variety of wheat was being used in 1935 when a new fungus (race 56) evolved which ruined the spring wheat. New rust-resistant varieties of wheat were used following this epidemic, but in 1953 and 1954 fungus race 15B evolved and attacked these varieties. Although even newer varieties of wheat are presently being used that are resistant to races 56 and 15B, wheat rust races are known that can attack these newer varieties as well.[118] Fungicides—such as copper salt mixtures, sulfur powder mixtures, organomercurials, organoarsenicals, and organozincs—are used extensively on crops.[90] Table 22 (Appendix) presents experimental data on the use of an eradicant fungicide.[80] Warning services are available for certain diseases—potato blight (Phytophthora infestans) for example—to tell farmers when they should spray with fungicides to control the spread of a disease. These warnings are based upon records of temperature and humidity or rainfall with consideration of the age of the crop and the susceptibility of the variety.[118] In recent years, aerial photography also has become a useful tool in the detection and control of crop diseases.[8] Sterilization or pasteurization of the soil is used when an area has become heavily infested with a pathogen. Heat, although expensive, has been and still is being used, but it is being replaced by chemicals—chloropicrin, Vapam, Mylone, formaldehyde, D-D mixture, and ethyl and methyl bromides.[14]

5. ECONOMICS

Ridker[94] has stated that because in many cases there are either insufficient or no data concerning the number of persons with a disease and very little information available concerning the cost of treatment, the economic loss due to the health effects of air pollutants is most difficult to estimate. The task is no less difficult with biological aerosols. One approach to the problem is to consider the incidence and prevalence of certain diseases. This will at least indicate the magnitude of the problem and the relative importance of the diseases.

One attempt at estimating a conservative dollar value for some diseases is presented in Table 6. The partial cost of tuberculosis is presented in Table 23, Appendix.

The influenza pandemic of 1918 to 1919 resulted in 550,000 deaths in the United States alone. It has been estimated that one-half of the world population suffered from the illness and that 20 million deaths occurred. The Asian flu pandemic of 1957 affected 45 million persons.[12]

As reported by the United States Bureau of the Census,[116] influenza and pneumonia ranked fifth as a cause of death in the United States in 1966, with an average rate of 32.5 deaths per 100,000 population. All other pulmonary diseases as a group were 10th in rank, with 14.5 deaths per 100,000 population (Table 24, Appendix). In 1966, the death rate for

TABLE 6

RESOURCE COSTS OF DISEASES ASSOCIATED WITH AIR POLLUTION[94]

	Costs Associated with Selected Diseases (Millions of Dollars)*						
Type of Cost	Cancer of the Respiratory System	Chronic Bronchitis	Acute Bronchitis	Common Cold	Pneumonia	Emphysema	Asthma
Premature Death	518	18	6		329	62	59
Premature Burial	15	0.7	0.2		13	2	2
Treatment	35	89		200	73		138
Absenteeism	112	52		131	75		60
Total	680	159.7	6.2	331	490	64	259

*Using a discount rate of 5 percent.

tuberculosis of all forms was 3.9, and that for meningococcal disease was 0.4 (Table 25, Appendix).

Data pertaining to the number of cases of specified reportable diseases in the United States are presented in Table 26, Appendix.

It has been estimated that people in the United States and Great Britain suffer from 2 to 10 acute respiratory illnesses each year.[115] The exact number of such illnesses reported is dependent upon the age of the person and his environment, and also on the number of symptoms and signs each investigator requires before he diagnoses a respiratory illness. The incidence, the number of days of restricted activity, and the number of days of bed rest for several respiratory diseases are presented in Table 27 (Appendix).[117] Table 28 (Appendix) shows the age distribution rates of certain reportable diseases.[28]

The control of plant diseases is a constant problem. Large epidemics among crops have occurred in the past. An epidemic of wheat rust in 1925 resulted in a loss of 12 million bushels of wheat, and another in 1935 in a loss of 135 million bushels.[107]

No information has been found on abatement and control costs pertaining to biological aerosols. However, the economic advantages of microorganisms in industrial fermentations are considerable. In antibiotic fermentation alone, the broad- and medium-spectrum antibiotics had a drugstore and hospital purchase cost of approximately 200 million dollars, and penicillin a purchase cost of 50 million dollars annually during the years 1959 to 1964.[58]

6. METHODS OF ANALYSIS

The problems of obtaining representative samples for analysis of airborne particles covering the wide range of atmospheric conditions, biological types, and particle size are such that no single procedure is adequate for all. Therefore, the methods of analysis tend to be specialized for relatively narrow fields of study; consequently, many different individual sampling devices have been used. The best reviews of the subject are by Wolf et al.,[127] Gregory,[46] Noble,[78] and May.[66]

The methods used for sampling biological aerosols are basically the same as the methods used to sample dust and other airborne particulates. However, since the objective is generally to determine the viability of collected particles, following collection the samples must undergo an additional step: growth in a suitable nutrient under proper environmental conditions, followed by observation of the growth and evaluation of the results.

Since no one method of analysis will yield information concerning all parameters of a sample, procedures should be chosen which will yield the information that is of greatest concern. The basic methods are these:

(1) Sedimentation:[93] In this method, particulates suspended in the air are allowed to settle either on plain surfaces or on surfaces coated with a nutrient medium. This method can yield information on the number of viable particles that have settled during the sampling time, and the total number and size of all particles that settle in a given time. Results will be influenced

greatly by air movement and diameter of the aerosol particles.

(2) Impingement into liquids:[19,37,43,75] Air is drawn through a small jet and is directed against a liquid surface, and the suspended particles are collected in the liquid. Due to the agitation of the particles in the collecting liquid, aggregates are likely to be broken up. Therefore, the counts obtained by this method tend to reflect the total number of individual organisms in the air and are higher than the values obtained by other methods.

(3) Impaction onto solid surfaces:[4,27] Air is drawn through a small jet(s), and the particles are deposited on dry or coated solid sufaces or on an agar nutrient. Samples taken by this method have been used to determine total numbers, size, viable numbers, and variation in numbers per unit of time during a long sampling period.

(4) Filtration:[72,79,101,111] The particulates are collected by passing the air through a filter, which can be made of cellulose-asbestos paper, glass wool, cotton, alginate wool, gelatin foam, or membrane material. The particulates are washed from the filters and assayed by appropriate microbiological techniques. In this method, the viability of organisms can be detrimentally affected by dehydration in the air stream and the results thereby biased.

(5) Centrifugation:[97,120] The particulates are propelled by centrifugal force onto the collecting surface, which can be glass or an agar nutrient. Size and number information can be obtained by this method.

(6) Electrostatic precipitation:[59] Particles are collected by drawing air at a measured rate over an electrically charged surface of glass, liquid, or agar. The total number of particles or viable number is then determined.

(7) Thermal precipitation:[57] Particles are collected by means of thermal gradients. The design is based on the principle that airborne particles are repelled by hot surfaces and are deposited on colder surfaces by forces proportional to the temperature gradient. The particle size distribution can be determined.

Because of the great number of different aerosol samples used by investigators, general agreement was reached at the International Aerobiology Symposium (sponsored by the Office of Naval Research and the University of California in October 1963) that data obtained with any specialized sampler should be correlated with at least some results obtained with a standard reference sampler.[7] The participants at the Symposium also agreed that the United States Army Chemical Corps all-glass impinger (AGI 30 Impinger)[127] be recommended as the standard liquid impinger, and that the Anderson Stacked Sieve sampler[4] be recommended as the standard apparatus for impaction on solid surfaces.

7. SUMMARY AND CONCLUSIONS

Biological aerosols—suspensions of microorganisms in the air—can cause diseases of humans, animals, and plants, and degradation of inanimate materials. The microorganisms generally involved are the bacteria, fungi (yeast and molds), and viruses. Bacterial and viral aerosols are detrimentally affected by the atmospheric environment and, therefore, airborne transmission of such diseases is limited to short distances and crowded conditions. Fungi are better adapted to aerial dissemination and are known to have been transmitted hundreds of miles from their source.

Generally, the symptoms produced by airborne infectious organisms in humans and animals are those of a respiratory disease. The human diseases in this category include tuberculosis, pneumonia, aspergillosis, influenza, the common cold, and others. As more data are gathered, there is increasing evidence that biological and nonbiological air pollutants are capable of producing synergistic effects. An increase in the incidence of respiratory diseases has been reported in metropolitan areas during occasions of excessively high air pollution. This potential effect has been confirmed through the use of experimental animals in the laboratory. For example, mice have been found to exhibit a higher mortality rate after a controlled dosage of Klebsiella pneumoniae when preceded by exposure to ozone or nitrogen dioxide.

Compared to humans, relatively few diseases of animals are spread by airborne transmission. Those that are include tuberculosis, glanders, aspergillosis, hog cholera, and Newcastle disease.

Plants are susceptible both to specific plant pathogens and to the indigenous saprophytic decay produced by microorganisms present in the soil. Of the plant pathogens, fungi are the most commonly transmitted by air and in the past have been the agents for devastating epidemics. For example, wheat rust destroyed an estimated 135 million bushels of wheat in 1935.

Saprophytic microorganisms are ubiquitous in nature. Consequently, surfaces of material in contact with a humid environment often show microbial—especially fungal—growth.

There are no environmental standards applicable to biological aerosols at the present time.

Sewage treatment plants have been investigated as a source of hazardous biological aerosols. Although potentially pathogenic microorganisms have been isolated downwind of sewage tanks, the full significance of this condition is not as yet known. Industrial fermentations with microorganisms produce a number of economically important materials—such as organic solvents, vitamins, and antibiotics—but no instance has yet been reported of a disease being transmitted to the general population as a result of any of these processes.

It is not valid to present any one value for the aerial microbial concentration of a given area. Any count is influenced by the temperature, meteorological conditions, vegetation, human and animal population, and time of day, as well as by the inability to determine all types of microorganisms by any one sampling procedure. However, some data have been presented as indicative of certain areas under noted environmental conditions and sampling procedures.

The problem of abatement and control of biological aerosols is exceedingly difficult and complex. Attempts have been made to control airborne infections indoors by ultraviolet light irradiation. Dust control, treatment of carriers with antibiotics, washing with disinfectant soaps, and the use of disinfectants and surgical masks have reduced significantly the spread of airborne disease in hospitals. Within recent years, a number of air filtration devices have been made commercially available that are capable of removing extremely small particles, including microorganisms. These devices have been produced in different sizes and efficiencies and can be used in air-conditioning systems. Their full potential in the control of biological aerosols nas not as yet been realized.

The control of outdoor airborne infections has been limited essentially to dust control and location and elimination of sources for specific outbreaks of certain diseases. Progress in the area has been hindered by lack of knowledge concerning the outdoor transmission of airborne disease.

There has been no adequate way to estimate the economic loss due to the effects of biological aerosols. However, the economic value of microorganisms in industrial fermentations is considerable.

The methods of analysis available for biological aerosols tend to be specialized for relatively narrow fields of study, and consequently many different individual sampling devices have been used. The basic methods are these: (1) sedimentation, (2) impingement into liquids, (3) impaction onto solid surfaces, (4) filtration, (5) centrifugation, (6) electrostatic precipitation, and (7) thermal precipitation.

Based on the material presented in this report, further studies are suggested in the following areas:

(1) More studies are needed to delineate the characteristics of biological aerosols with the goal of better understanding their production, survival, and dispersal in indoor and outdoor areas. For example, what is the relative significance of the transmission of a disease—such as influenza—outdoors as compared to indoors?

(2) Further documentation of the synergistic effects of biological and nonbiological air pollutants is warranted.

(3) Additional information is needed on the value of upgrading air conditioning systems with filters and ultraviolet light in schools, office areas, and other places for control of biological aerosols.

(4) Further delineation of the potential sources of hazardous biological aerosols is necessary.

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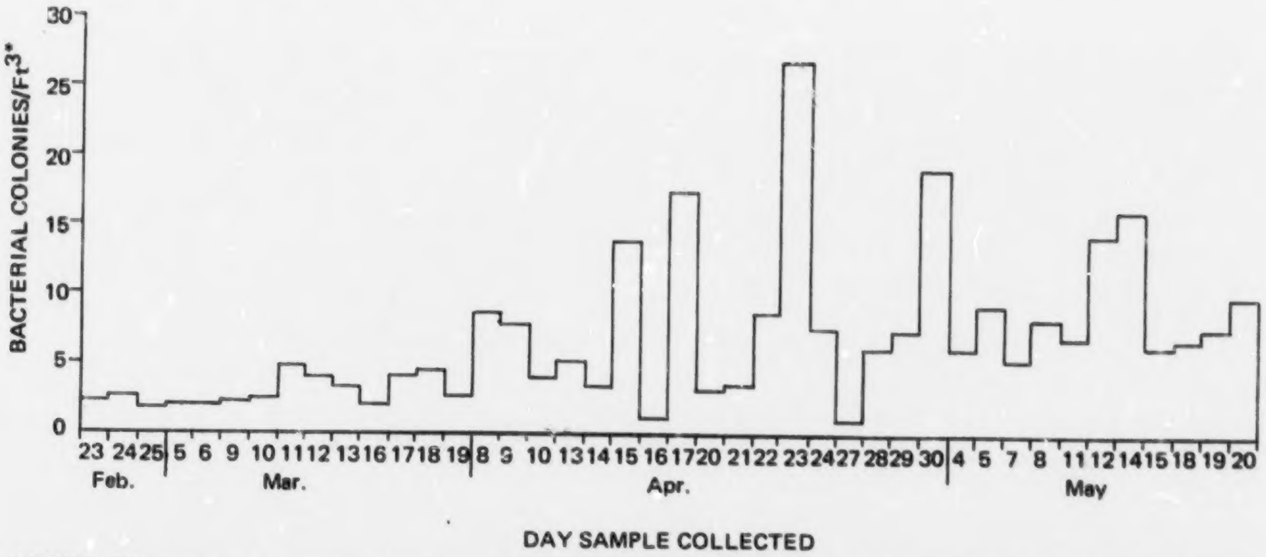
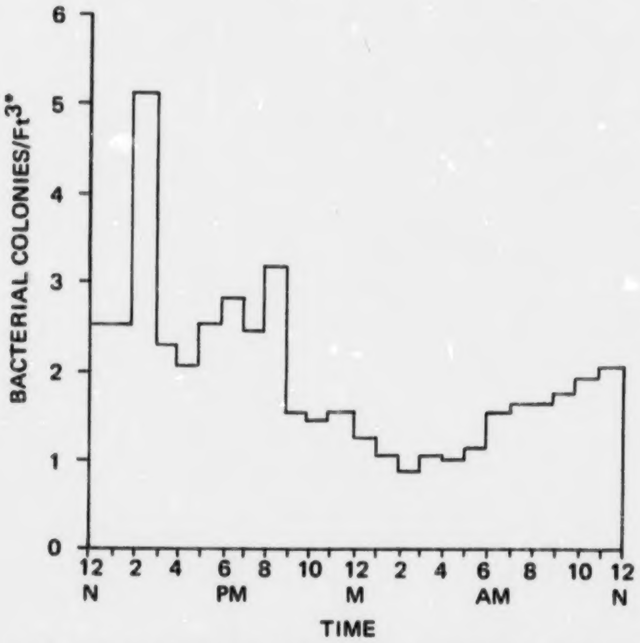
APPENDIX



*NOTE: Extramural, sieve sampler, heart infusion agar with 5 percent blood added, 37°C incubation for 48 hours, Feb.-May 1953, Detroit, Mich.

FIGURE 1

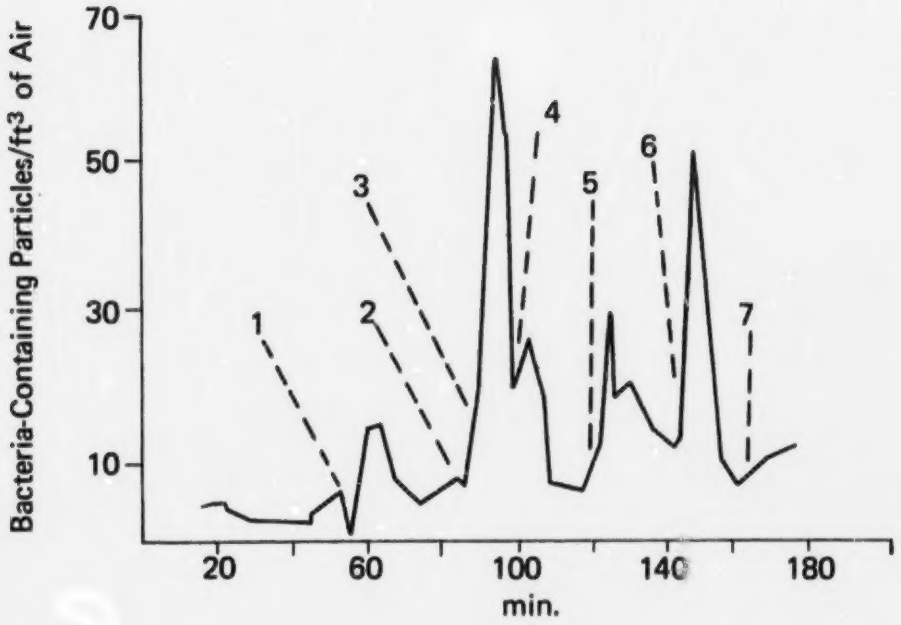
Daily Bacterial Counts in Urban Area[127]



*NOTE: Extramural, sieve sampler, heart infusion agar with 5 percent blood added, 37°C incubation for 24 hours, June 27-July 3, 1951, Oatland Island, Savannah, Ga.

FIGURE 2

Hourly Bacterial Counts in Nonurban Area[127]



1 - 1st patient in
2 - 2nd patient in
3 - Patient rolled over by 5 people
4 - Table moved by 5 people
5 - Patient moved on table
6 - Patient rolled back
7 - 3rd patient in

FIGURE 3

Airborne Organisms in a Surgery Room[22]

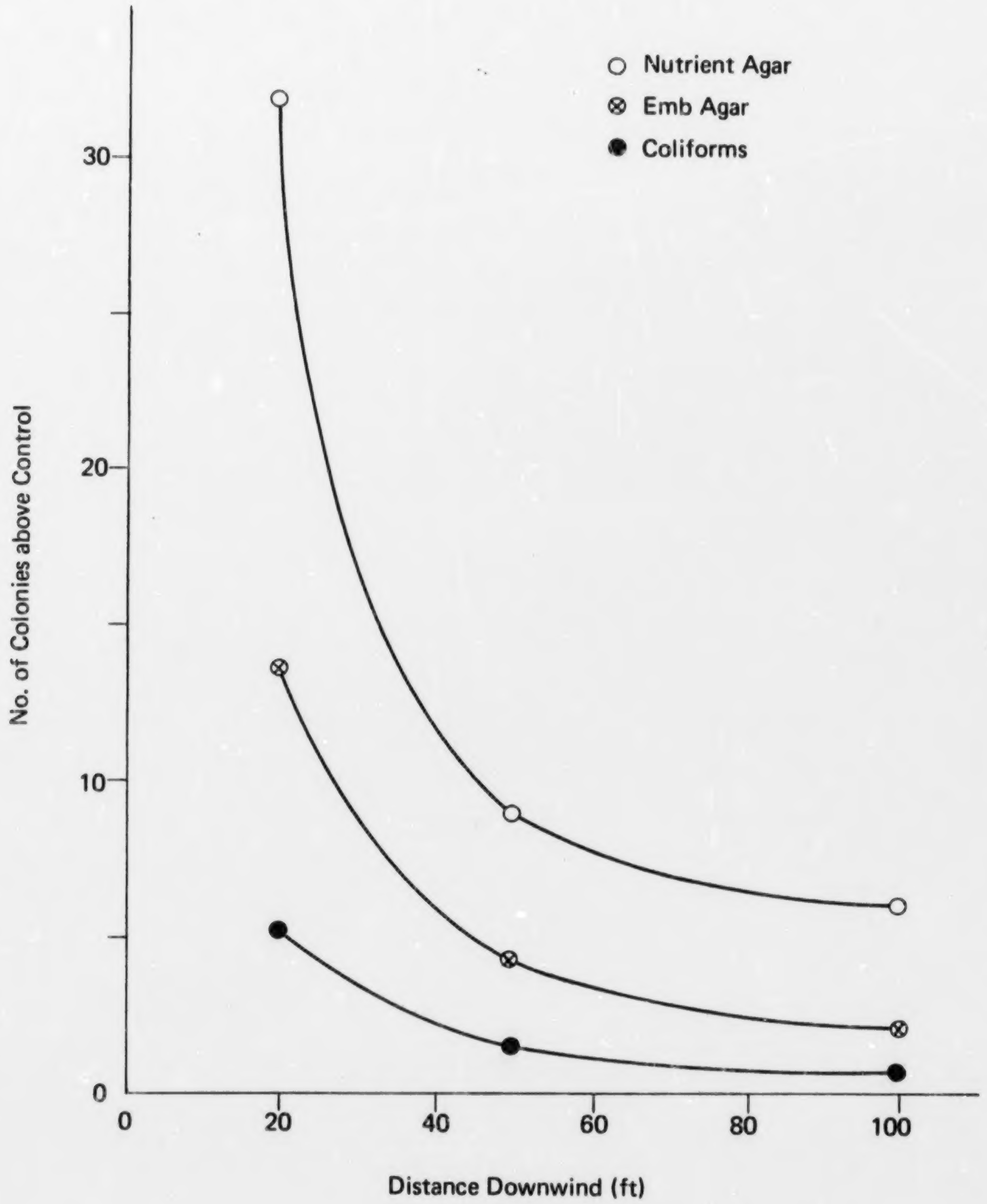
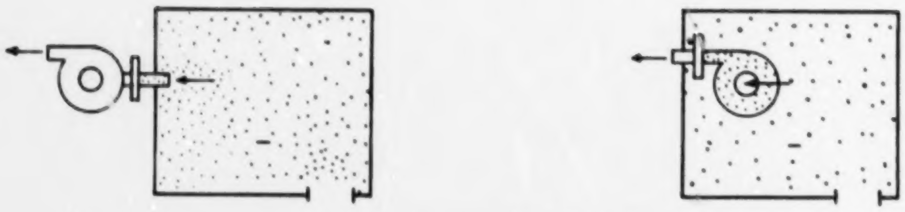
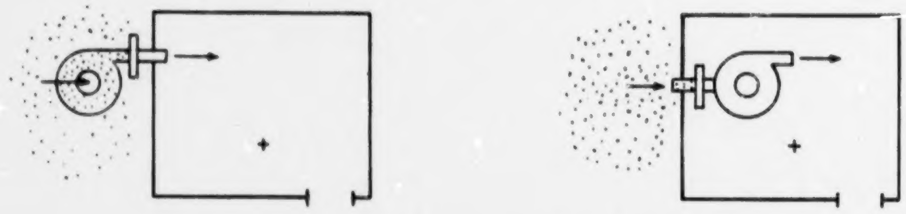


FIGURE 4

Effect of Distance Downwind of Treatment Unit[62]



A. Contamination Within Room
Inside Pressure Should Be Lower Than Outside



B. Contamination Outside Room
Inside Pressure Should Be Greater Than Outside

FIGURE 5

Relative Position of Filter and Blower to Confine Contamination Inside or Outside Room[23]

APPENDIX

TABLE 7

COMMON AIRBORNE BACTERIAL INFECTIONS OF HUMANS[12,17,38,95,105,112,114]

Disease	Causative Agent	Symptoms and Remarks
Pulmonary tuberculosis	*Mycobacterium tuberculosis*	Lesions caused by nodules or tubercles are found in the lungs (or other parts of the body). In some cases calcification of the nodules takes place, and in others there is a coalescence of the necrotic tissue
Pulmonary anthrax	*Bacillus anthracis*	Primarily a disease of animals but also occurs in man. This is the most dangerous, although not the most common, of the three forms of anthrax. It is characterized by many of the symptoms of pneumonia and often progresses into fatal septicemia
Staphylococcal respiratory infection	*Staphylococcus aureus*	Can result in a gradual cavitating pneumonia or a fulminating hemorrhagic pneumonia
Streptococcal respiratory infection	*Streptococcus pyogenes*	May develop into any of a variety of symptoms, including tonsillitis, sinusitis, otitis media, bronchopneumonia, pharyngitis, or septic sore throat, and becomes scarlet fever if the infecting strain produces erythrogenic toxin

(continued)

TABLE 7 (Continued)

COMMON AIRBORNE BACTERIAL INFECTIONS OF HUMANS

Disease	Causative Agent	Symptoms and Remarks
Meningococcal infection	*Neisseria meningitidis*	Probably becomes established initially in the nasopharynx but clinically develops into a cerebrospinal meningitis
Pneumococcal pneumonia	*Diplococcus pneumoniae*	Clinically is nearly always lobar pneumonia. However, the infection may migrate through the nasal passages or be distributed via the vascular system to various parts of the body and give rise to localized foci of infection. Death is due to overwhelming interference with respiration or to general systemic toxemia
Pneumonic plague	*Pasteurella pestis*	Although ordinarily spread by the bite of fleas, it can occur secondary to glandular plague and give rise to a primary pulmonary form transmitted from man to man; usually fatal

(contined)

TABLE 7 (Continued)

COMMON AIRBORNE BACTERIAL INFECTIONS OF HUMANS

Disease	Causative Agent	Symptoms and Remarks
Whooping cough	*Bordetella pertussis*	Usually a childhood disease which begins with a catarrhal stage of a mild cough that progresses in severity to a paroxysmal stage characterized by rapid consecutive coughs and the deep inspiratory whoop. In the convalescent stage, the number and frequency of paroxysms gradually decrease
Diphtheria	*Corynebacterium diphtheriae*	A childhood disease, usually a local infection of the mucous surfaces. The pharynx is most commonly affected, but infection of the larynx, or membranous croup, and nasal diphtheria are not infrequently observed. Primary infection of the lungs and other parts of the body have been reported
Klebsiella pulmonary infection	*Klebsiella pneumoniae*	Produces necrotic lesions of the lung parenchyma and usually is fatal if not treated

(continued)

TABLE 7 (Continued)

COMMON AIRBORNE BACTERIAL INFECTIONS OF HUMANS

Disease	Causative Agent	Symptoms and Remarks
Staphylococcal wound infection	Staphylococcus aureus	Those surgical wounds which become infected by bacteria settling from air in the surgery room. These organisms may be derived from the surgical team or may be carried into the operting room by air currents

APPENDIX

TABLE 8

COMMON AIRBORNE FUNGAL INFECTIONS OF HUMANS[12,17,38,95,105,112,114]

Disease	Causative Agent	Symptoms and Remarks
Blastomycosis	*Blastomyces dermatitidis*	A chronic granulomatous mycosis clinically resembling tuberculosis with coughing, pain in the chest, and weakness
Coccidioidomycosis	*Coccidioides immitis*	Varies in severity in recognized primary cases from that of a common cold to cases resembling influenza. Many cases are symptomless. The secondary or progressive coccidioidomycosis results in cutaneous, subcutaneous, visceral, and osseous lesions with a high fatality rate
Cryptococcosis	*Cryptococcus neoformans*	More commonly is a generalized infection, but can also be a primary (or secondary) lung infection. It may spread from the lungs as well
Histoplasmosis	*Histoplasma capsulatum*	A systemic mycosis of varying severity, with the primary lesion usually in the lungs. Clinical symptoms of the systemic form can resemble many other diseases (anemia, leukopenia, Hodgkin's disease, etc.)

(continued)

APPENDIX

TABLE 8 (Continued)

COMMON AIRBORNE FUNGAL INFECTIONS OF HUMANS

Disease	Causative Agent	Symptoms and Remarks
Nocardiosis	*Nocardia asteroides*	A chronic disease resembling tuberculosis, often initiated in the lungs but sometimes progressing to a systemic infection
Aspergillosis	*Aspergillosis fumigatus*	A chronic pulmonary mycosis similar to and sometimes mistaken for tuberculosis. The infection may be secondary, particularly to tuberculosis. Pulmonary infection results from inhalation of airborne spores
Sporotrichosis	*Sporotichum schenckii*	A nodular skin infection ultimately forming a necrotic ulcer. Transmission by inhalation of spores is rare

APPENDIX

TABLE 9

VIRAL AND RELATED AGENTS PRESENTLY RECOGNIZED AS THE CAUSE OF HUMAN RESPIRATORY DISEASES[51]

Group	Number Serotypes Causing Respiratory Illness	Serotype Name	Types of Clinical Syndromes Produced	Comments
1. Myxoviruses	2	Influenza A	Influenza, febrile pharyngitis or tonsillitis, common cold, croup, bronchitis, bronchiolitis, pneumonia	Causes influenza in persons of all ages
		Influenza B		
	?	Influenza C		
	1	Respiratory Syncytial (RS)	Bronchiolitis (infants), pneumonia, bronchitis, common cold, croup	Most common cause of bronchiolitis in children
	4	Parainfluenza	Croup (infants), bronchitis, common cold, pneumonia, bronchiolitis	Type 1 is the most important agent in the croup syndrome
2. Adenoviruses	8	1, 2, 3, 4, 5, 7, 4, 21	Bronchitis, common cold, pneumonia, brochiolitis, febrile sore throat	

(continued)

TABLE 9 (Continued)

VIRAL AND RELATED AGENTS PRESENTLY RECOGNIZED AS THE CAUSE OF HUMAN RESPIRATORY DISEASES

Group	Number Serotypes Causing Respiratory Illness	Serotype Name	Types of Clinical Syndromes Produced	Comments
3. Picornaviruses	7	Coxsackie A (2, 3, 5, 6, 8, 10, 21)	Febrile sore throat, common cold	
	3	Coxsackie B (2, 3, 5)	Febrile sore throat, common cold, pleurodynia	
	60+	Rhinoviruses	Common cold, bronchitis, pneumonia	Most frequently isolated viruses in adults with upper respiratory infections
	2	ECHO (11, 20)	Febrile sore throat, common cold, croup	
3a. Reoviruses (classification uncertain)	3	Reovirus (ECHO-10)	Minor respiratory symptoms and diarrhea (children)	
4. Herpesviruses	3	Herpes	Pharyngitis (adults)	
	1	Varicella	Pneumonia	

(continued)

TABLE 9 (Continued)

VIRAL AND RELATED AGENTS PRESENTLY RECOGNIZED AS THE CAUSE OF HUMAN RESPIRATORY DISEASES

Group	Number Serotypes Causing Respiratory Illness	Serotype Name	Types of Clinical Syndromes Produced	Comments
5. Chlamydozoaceae*	?	Psittacosis	Psittacosis, pneumonia	
6. Mycoplasmataceae	1	*Mycoplasma pneumoniae*	Pneumonia (Eaton agent), bronchitis, bronchiolitis, minor upper respiratory illness	
7. Rickettsiae		*Coxiella burnetii* (Q fever)	Pneumonia	

*Not a true virus; nucleic acid core contains both RNA and DNA.

APPENDIX

TABLE 10

POSSIBLE AIRBORNE VIRUS DISEASES OF ANIMALS[11,69,70]

Disease	Host	Symptoms and Effects	Morbidity and Mortality	Control
Hog cholera	Swine	Fever, stilted gait, conjunctivitis, diarrhea	As high as 90% mortality	Immunization
Equine influenza	Horse	Fever, nasal discharge, abortion in mares	Low mortality	Immunization
Swine influenza	Swine	Exudative bronchitis	Morbidity almost 100%, mortality 2% or less	None
Feline distemper	Cat, mink, raccoon	Vomiting, diarrhea, nasal and eye discharge	Recovery usual	Immunization
Canine distemper	Dog, fox, mink	Fever, diarrhea, rhinitis	Recovery usual	Immunization
Newcastle disease	Chicken, turkey, ducks, other fowl	Coughing, sneezing, paralysis of legs, loss of egg production	Morbidity 100%, mortality 5-50%	Immunization
Infectious bronchitis	Chicken	Rales, wheezing, loss of egg production	Mortality up to 60% in chicks, neglible in older birds	Immunization

APPENDIX

TABLE 11

COMMON LABORATORY ANIMALS USED IN STUDIES OF AIRBORNE DISEASE[1,54,114]

Disease	Laboratory Animal								
	Mouse	Guinea Pig	Rabbit	Rat	Monkey	Cat	Dog	Pigeon	Chicken
Pulmonary tuberculosis	x	x	x		x				
Pulmonary anthrax	x	x	x		x				
Staphylococcal respiratory infection			x						
Streptococcal respiratory infection	x	x	x						
Meningococcal infection*									
Pneumococcal pneumonia	x	x	x			x	x		
Pneumonic plague		x			x				
Whooping cough	x	x	x						
Diphtheria		x				x	x		x
Pulmonary Klebsiella infection	x	x	x						
Staphylococcal wound infections	x	x	x	x	x				
Aspergillosis			x					x	
Blastomycosis	x	x							
Coccidioidomycosis	x	x	x		x				
Cryptococcus	x								
Histoplasmosis	x	x	x	x			x		
Nocardiosis		x							

*Induced with difficulty or unsuccessfully in laboratory animals.

APPENDIX

TABLE 12

AVERAGE MICROPOPULATION PER CUBIC METER FOUND SIMULTANEOUSLY DURING 30-HOUR SAMPLING MISSION[39]

Altitude (meters)	Time			
	0600-1200	1200-1800	1800-2400	0000-0600
690	45	250	200	90
1,600	25	65	75	50
3,127	23	30	35	15

TABLE 13

QUANTITATIVE RESULTS FROM THE BALLOON-BORNE DIRECT-FLOW SAMPLERS[10]

Altitude (thousand feet)	Average Volume (ft^3 air/microbe)
10-30	50-100
30-60	330-500
60-90	2,000

TABLE 14. AIR DISPERSION OF SMALL ORGANISMS[3]

Disease (Organism)	Means of Dispersion	Distances and Units Dispersed (Horizontal Dispersion)					
(Airborne spores)	Wind	Degrees north of equator Fungus colonies on plate	$57^{\circ}30'$ 3.61	$64^{\circ}20'$ 0.49	$68^{\circ}55'$ 0.48	$71^{\circ}5'$ 0.72	
Beet downy mildew (*Peronospora* sp.)	Wind	Meters from seed plants Plants injured, %	10 28	150 8	1,000 1		
Blossom infection (*Sclerotinia laxa*)	Air currents	Feet from center of nearest source row Blossom infection, %	22 55.7	44 39.1	66 29.3	68 22.4	
(*Bovista plumbea*)	Air currents	Meters from release point Spores caught	5 912	10 323	15 165	20 102	
Cedar and apple rust (*Gymnosporangium* sp.)	Air currents	Yards from infected trees Leaf infections	0 64	55 40	110 33	220 26	440 19
Chestnut blight (*Endothia parasilica*)	Air currents	Feet from spore source Ascospores found	27 23	85 11	180 8	266 8	
Crown rust of oats (*Puccinia coronata*)	Wind	Feet from inoculum source Infections, %	3 92.9	5 53.4	7.7 35	10.3 19.5	13 0.7
Downy mildew (*Pseudoperonospora humuli*)	Air currents	Feet from spore source Leaves infected, %	10 26	50 16	100 12	200 7	400 3
Leaf spots on tulips	Raindrop splash and wind	Centimeters from conidia source Lesions/plant	15.2 31.6	34.6 20.1	58.0 12.9	79.8 8.5	102.0 5.1
Loose smut of wheat (*Ustilago tritici*)	Air currents	Meters from spore source Smutted heads	2 241	4 234	24 114	80 0	
Maize rust (*Puccinia sorghi*)	Wind	Kilometers from spore source Plants attacked, %	0.5 100	2.5 3	4.5 0.3	6.5 0	
Onion mildew (*Peronospora destructor*)	Air currents	Feet from onion sets Lesions/100-ft row	120 1,138	780 98	1,750 1	2,000 0	

(continued)

TABLE 14. AIR DISPERSION OF SMALL ORGANISMS (Continued)

Disease (Organism)	Means of Dispersion	Distances and Units Dispersed (Horizontal Dispersion)					
Potato late blight (Phytophthora infestans)	Wind	Centimeters from edge of infective group Plants infected, %	30 89	90 63	150 43	210 22	270 5
Powdery mildew on barley (Erysiphe graminis)	Wind	Meters from source Plants affected, %	1.5 99	3.5 84	5.5 76	7.5 70	8.5 68
Stem rust (Puccinia graminis)	Wind	Feet from barberry hedge Grass infected, %	15 100	125 41	225 5	325 1	425 0.5
Stem rust on rye (P. graminis secalis)	Wind	Meters from source plant g/100 ears	50 47.6	300 92.3	1,000 122.3	3,000 149.7	
(Tilletia tritici)	Air currents	Meters from release point Spores caught	5 800	10 168	15 49	20 30	
Tobacco blue mold (Peronospora tabacina)	Wind	Yards from source Plant lesions/1,000 in^2 of field	0 140	4 8	8 1	12 0.5	
Wheat stem rust (Puccinia graminis)	Air currents	Miles from known source Spores collected	200 13,092	360 10,768	580 8,883	740 7,920	940 6,975
White pine blister rust (Cronartium ribicola)	Air currents	Feet from gooseberry bush Diseased trees, %	50 75	150 55	350 40	450 36	650 29
		(Vertical Dispersion)					
Azalea flower spot (Ovulinia azaleae)	Air currents	Inches above ground Infections	4 42	10 28	18 17	48 0	
Onion mildew (Peronospora destructor)	Air currents	Altitude, feet Spores/ft^3 air	100 32	200 102	700 451	1,200 801	

(continued)

TABLE 14. AIR DISPERSION OF SMALL ORGANISMS (Continued)

Disease (Organism)	Means of Dispersion	Distances and Units Dispersed (Vertical Dispersion)					
Wheat stem rust (*Puccinia graminis*)	Air currents	Feet above barberry bushes	1,000	2,000	7,000	12,000	
		Aeciospores caught	19	14	5	1	
		Altitude, feet	1,000	5,000	10,000	14,000	
		Urediospores	48,200	7,730	144	40	
		Elevation, meters	30	400	600	800	
		Spores/cm^2/min	1,458	490	339	231	

APPENDIX

TABLE 15

RECOMMENDED CONDITIONS FOR USE OF COMMON GERMICIDAL SUBSTANCES AT ROOM TEMPERATURE (25° C)[54]

Germicide	Concentration and Exposure Time for Typical Classes of Microorganisms			
	Vegetative Bacteria	Bacterial Spores	Fungi	Bacterial Toxins
Phenol	5% (5 min)	NR[a]	5% (15 min)	NR[a]
Lysol	2% (5 min)	NR[a]	3% (15 min)	NR[a]
Quaternary ammonium compounds (Roccal, Purasan, Hyamine, etc.)	0.1-1.0% (5 min)	NR[a]	NR[a]	NR[a]
Hypochlorites + 1% wetting agent (Naccanol, etc.)	200-1,000 ppm (1 min)	500-5,000 ppm (5 min)	2,000 ppm (10 min)	NR[a]
Caustic sodium hydroxide	2% (15 min)	5% (30 min)	10% (30 min)	5% soln (pH 11.5) (15 min)
Formalin (37% HCHO)	5% soln (10 min)	10% soln (10 min)	5% soln (10 min)	5% soln (10 min)
Steam formaldehyde vapor (closed areas)	1 ml/ft^3 in air with RH[b] above 80% (30 min)			NR[a]
beta-Propiolactone vapor	200 mg/ft^3 in air with RH[b] above 80% (30 min)			NR[a]
Ethylene oxide gas	300 mg/liter (8-16 hr)			NR[a]

[a] NR = not recommended.

[b] RH = relative humidity.

APPENDIX

TABLE 16

MATHEMATICAL MODEL ON HOSPITAL VENTILATION[23]

Let

N = number of organisms/ft^3 present at time t in minutes

V = volume of room in cubic feet

K = number of complete changes of room volume/hour

b = total number of organisms/minute entering because of human presence

a = efficiency of the filter

Then,

$\frac{NKV}{V60}(1-a)\,\Delta t$ = total number of organisms/ft^3 entering the interval Δt because of the inefficiency of the filter.

$\frac{1}{V}\,b\,\Delta t$ = total number of organisms/ft^3 entering during interval Δt because of contamination from individuals.

$\frac{1}{V}\,\frac{NKV}{60}\,\Delta t$ = total number of organisms/ft^3 leaving during Δt.

ΔN = (total number of organisms/ft^3 entering) - (total number of organisms/ft^3 leaving)

$$\Delta N = \frac{NK}{60}(1-a)\,\Delta t + \frac{b}{V}\,\Delta t - \frac{NK}{60}\,\Delta t$$

$$\frac{\Delta N}{\Delta t} = \frac{b}{V} - \frac{KNa}{60}$$

$$\frac{dN}{dt} = \frac{b}{V} - \frac{KaN}{60} = \frac{b}{V}\left\{1 - \frac{aKVN}{60b}\right\}$$

$$\frac{dN}{1-\frac{aKVN}{60b}} = \frac{b}{V}\,dt$$

$$-\frac{60b}{aKV}\int\frac{\frac{-aKVdn}{60b}}{1-\frac{aKVN}{60b}} = \int\frac{b\,dt}{V}$$

(Continued)

APPENDIX

TABLE 16 (Continued)

MATHEMATICAL MODEL ON HOSPITAL VENTILATION

$$-\frac{60b}{aKV}\ \mathrm{In}\ \left\{1-\frac{aKVN}{60b}\right\}=\frac{b}{V}\,t+C$$

If $t = 0$
$N = 0$
then, $C = 0$

$$-\frac{60b}{aKV}\ \mathrm{In}\ \left\{1-\frac{aKVN}{60b}\right\}=\frac{b}{V}\,t$$

$$-\frac{60}{aK}\ \mathrm{In}\ \left\{1-\frac{aKVN}{60b}\right\}=t$$

$$\mathrm{In}\ \left\{1-\frac{aKVN}{60b}\right\}=-\frac{aKT}{60}$$

$$\left\{1-\frac{aKVN}{60b}\right\}=\exp-\frac{aKt}{60}$$

$$\frac{60b}{aKV}\left[1-\exp\left\{-\frac{aKt}{60}\right\}\right]=N$$

TABLE 17

ROUGHING FILTERS[23]

(Particle Retention[a] 10 to 60 Percent[b])

Nomenclature	Manufacturer	Media	Capacity cfm/ft^2 of Face A	Face Velocity (ft/min)	Pressure Drop (H_2O)	Maximum operation temperature
AAF type HV 2	American Air Filter Corp., Louisville, Ky.	Adhesive-coated V-crimped wire screen mesh	250 to 430	300 to 500	0.004"	110°F
AAF PL24 with type G media	American Air Filter Corp.	Glass filament	up to 250	250	0.06"	250°F
Drico puff-glass	Drico Industrial Corp. Passaic, N.J.	Spun glass fiber	32 to 1,000	300	0.08" to 0.11"	175°F
Farr-Air HP-2	Farr Filter Co., Los Angeles, Calif.	Pleated cotton fabric	250 to 435	250 to 435	0.045" to 0.115"	255°F
Farr 44-68	Farr Filter Co.	Crimped screen and wire mesh	250 to 435	250 to 435	0.040"	275°F

[a] One to five μ

[b] Inclusion of any particular filter in this table does not constitute endorsement by the United States Government or by the authors.

TABLE 18

MEDIUM-EFFICIENCY FILTERS[23]
(Particle Retention[a] 60 to 90 Percent[b])

Nomenclature	Manufacturer	Media	Capacity cfm/ft^2 of Face A	Face Velocity (ft/min)	Pressure Drop (H_2O)	Maximum operation temperature
AAF deep bed Type 100 FG	American Air Filter Corp., Louisville, Ky.	Fiberglass	50 to 250	250	0.24"	700°F
AAF PL 24 frame Type 25 FG	American Air Filter Corp.	Fiberglass	50 to 250	200	0.09"	400°F
Aerosolve 45	Cambridge Filter Corp., Syracuse, N.Y.	Glass fibers	up to 500	250 to 500	0.16" to 0.25"	400°F
Expandure	Flanders Filters, Riverhead, N.Y.	Fiberglass	250	250	0.38"	200°F

(continued)

TABLE 18 (Continued)

MEDIUM-EFFICIENCY FILTERS[23]
(Particle Retention[a] 60 to 90 Percent[b])

Nomenclature	Manufacturer	Media	Capacity cfm/ft^2 of Face A	Face Velocity (ft/min)	Pressure Drop (H_2O)	Maximum operation temperature
Type CA	Microtron Corp., Charlotte, N.C.	Polyester/ acetate adhesive-coated	200 to 250	200 to 250	0.08" to 0.13"	350°F
U-Lok	Union Carbide Development Co., N.Y., N.Y.	Dynel fibers	200 to 500	300	0.10"	180°F

[a] One to five μ.

[b] Inclusion of any particular filter in this table does not constitute endorsement by the United States Government or by the authors.

TABLE 19

HIGH-EFFICIENCY FILTERS[23]

(Particle Retention[a] 90 to 99 Percent[b])

Nomenclature	Manufacturer	Media	Capacity cfm/ft^2 of Face A	Face Velocity (ft/min)	Pressure Drop (H_2O)	Maximum operation Temperature
Multi-Pak[c] with 50 FG	American Air Filter Corp., Louisville, Ky.	Glass fiber	125 to 250	250	0.42"	400°F
Deep bed with 50 FG	American Air Filter Corp.	Glass fiber	40 to 200	200	0.42"	400°F
Micretain	Cambridge Filter Corp. Syracuse, N.Y.	Glass-asbestos pleated	50 to 250	Up to 250	0.4"	220°F to 800°F
Aerosolve 85	Cambridge Filter Corp.	Glass fibers pleated	125 to 500	250 to 500	0.22" to 0.32"	400°F
Aerosolve 95	Cambridge Filter Corp.	Glass fiber pleated	125 to 500	250 to 500	0.35" to 0.45"	400°F

(continued)

TABLE 19 (Continued)

HIGH-EFFICIENCY FILTERS
(Particle Retention[a] 90 to 99 Percent[b])

Nomenclature	Manufacturer	Media	Capacity cfm/ft^2 of Face A	Face Velocity (ft/min)	Pressure Drop (H_2O)	Maximum Operation Temperature
HP-100	Farr Filter Co., Los Angeles, Calif.	Glass fiber pleated	250	250	0.20"	275°F
HP-200	Farr Filter Co.	Glass fiber	250	250	0.38"	275°F

[a] One to five μ.

[b] Inclusion of any particular filter in this table does not constitute endorsement by the United States Government or by the authors.

[c] These filters made to accommodate double thickness of media.

TABLE 20

ULTRA-HIGH EFFICIENCY FILTERS[23]
(Particle Retention[a] More than 99.99 Percent[b])

Nomenclature	Manufacturer	Media	Capacity[c] cfm/ft^2 of Face A	Face Velocity[d] ft/min	Pressure Drop (H_2O)	Maximum Operation Temperature
AAF Type F (glass)	American Air Filter Corp., Louisville, Ky.	Glass fiber and kraft paper or alum sep.	30 to 400	68 to 325	1.0"	250°F to 1,000°F
AAF Type F (ceramic)	American Air Filter Corp.	Ceramic asbestos fiber and alum sep.	30 to 250	250	1.0"	1,600°F to 2,300°F
Cambridge Absolute	Cambridge Filter Corp., Syracuse, N.Y.	Glass fiber asbestos paper sep.	30 to 345	Up to 275	1.0"	800°F
Magnamedia	Farr Filter Co., Los Angeles, Calif.	Glass fiber	30 to 400	Up to 250	1.0"	Up to 1,000°F
Airpure absolute glass F 600	Flanders Filters, Riverhead, N.Y.	Glass fiber (F 600)	30 to 400	Up to 320	1.0"	850°F

(continued)

TABLE 20 (Continued)

ULTRA-HIGH EFFICIENCY FILTERS

(Particle Retention[a] More than 99.99 Percent[b])

Nomenclature	Manufacturer	Media	Capacity[c] cfm/ft^2 of face A	Face Velocity[d] ft/min	Pressure Drop (H_2O)	Maximum Operation Temperature
Airpure absolute ceramic-asbestos	Flanders Filters	Ceramic-asbestos	50 to 250	Up to 250	1.0"	1,600°F
Ultra-Aire	Mine Safety Appliance Co. Pittsburgh, Pa.	Glass fiber	35 to 250	Up to 250	0.9"	500°F

[a] One to five μ.

[b] Inclusion of any particular filter in this table does not constitute endorsement by the United States Government or by the authors.

[c] Capacities are in cfm/ft^2 of face area, not total area of filter.

[d] Face velocities are fpm for 1 ft^2 of face area, not media velocity.

APPENDIX

TABLE 21

PENETRATION OF T1 PHAGE[a] AND BACTERIAL AEROSOLS[b] THROUGH COMMERCIAL AIR FILTERS[48]

Filter type	Description	Test Number	Relative Humidity %	Test Air Flow	Filter Resistance (water)	Penetration		
						T1 Phage[c] %	Bacterial Spores[d] %	DOP[e] %
Ultrahigh-efficiency	Glass microfibers waterproofed, plastic base adhesive, 35 cfm rated capacity 8" x 8 " x 3-1/16"	1	15 to 20	25 cfm	1.04"	3.2×10^{-3}	8.7×10^{-5}	0.011
		2				4.3×10^{-3}	9.6×10^{-5}	
		3				4.3×10^{-3}	1.4×10^{-4}	
		Mean				3.9×10^{-3}	1.1×10^{-4}	
Ultrahigh-efficiency	Glass asbestos fibers with organic binder, neoprene type sealer, 30 cfm rated capacity 8" x 8" x 3-1/16"	1	15 to 20	25 cfm	0.69"	1.2×10^{-3}	8.4×10^{-5}	0.02
		2				6.0×10^{-4}	6.1×10^{-5}	
		3				7.6×10^{-4}	7.2×10^{-5}	
		Mean				8.5×10^{-4}	7.2×10^{-5}	
Ultrahigh-efficiency	All-glass fibers with no organic binder, rubber base type sealer, 30 cfm rated capacity 8" x 8" x 3-1/16"	1	20 to 25	25 cfm	0.53"	4.6×10^{-3}	4.0×10^{-4}	0.006
		2				3.9×10^{-3}	1.7×10^{-4}	
		3				4.7×10^{-3}	2.8×10^{-4}	
		Mean				4.4×10^{-3}	2.8×10^{-4}	

(continued)

APPENDIX

TABLE 21 (Continued)

PENETRATION OF T1 PHAGE[a] AND BACTERIAL AEROSOLS[b] THROUGH COMMERCIAL AIR FILTERS

Filter Type	Description	Test Number	Relative Humidity %	Test Air Flow	Filter Resistance (water)	Penetration		
						T1 Phage[c] %	Bacterial Spores[d] %	DOP[e]
Ultrahigh-efficiency	All-glass fibers with no organic binder, rubber base type sealer, 22 cfm rated capacity 8" x 8" x 12"	1	15 to 20	22 cfm	0.75"	$1.1\text{x}10^{-3}$	$1.9\text{x}10^{-3}$	0.002
		2				$1.0\text{x}10^{-3}$	$2.2\text{x}10^{-3}$	
		3				$9.9\text{x}10^{-4}$	$2.8\text{x}10^{-3}$	
		Mean				$1.0\text{x}10^{-3}$	$2.3\text{x}10^{-3}$	
Over-all mean for ultrahigh-efficiency filter units						$3\ \text{x}10^{-3}$	$7\ \text{x}10^{-4}$	
High efficiency	0.5" thick fiberglass pads containing 1.25 μ diameter glass fibers	1	40 to 45	20ft per min[f]	0.50	1.8	0.23	
		2			0.50	2.0	0.26	
		3			0.51	1.9	0.50	
		Mean				1.9	0.33	

[a] T1 phage aerosol number median diameter (NMD): 0.1 u.

[b] B. subtilis var. niger spore aerosol NMD: 1 u.

[c] Prefilter total sampler (impinger + backup filter) recovery: 10^8 phage/liter.

[d] Prefilter cotton collector recovery: 10^5 spores/liter.

[e] DOP penetration as stamped on filter unit by manufacturer.

[f] Face velocity (1.5 cfm through 3-3/4 inch diameter filter pads).

APPENDIX

TABLE 22

EFFECT OF ERADICANT FUNGICIDES ON SPORODOCHIA PRODUCTION, CONIDIAL GERMINATION, AND BLOSSOM BLIGHT CAUSED BY MONILIA LAXA ON DRAKE ALMOND, 1958[80]

Fungicide	Dates of application	Average number of sporodochia per twig	Twigs with sporodochia (%)	Conidial germination on agar (%)	Amount blossom blight per 100 20-inch shoots inspected
		Orchard No. 1			
SPCP[a]	12/12/57	0.58	18	94[b]	21.2
SPCP plus LLS[a]	12/12/57	0.94	16	26	15.7
SPCP	1/22/58	0.28	20	34	42.4
Untreated		2.04	74	78	93.1
		Orchard No. 2			
SPCP	12/13/57	4.6	100	46[c]	60.9
SPCP plus LLS	12/13/57	1.7	48	1	37.8
SPCP	1/9/58	8.8	76	3	78.1
Untreated		14.7	96	60	232.0

[a] SPCP is 8.0 pounds of 37% sodium pentachlorophenoxide in 100 gallons of water applied at the rate of 400 gal/acre with an airblast sprayer, and LLS is 11.2 gal of 32° Baume calcium polysulfide combined with SPCP.

[b] Potato dextrose agar.

[c] Water agar.

APPENDIX

TABLE 23

TUBERCULOSIS HOSPITAL USE[116]
(Rates per 1,000 Population)

Year	Admission Rate	Total Days in Hospital	Average Length of Stay (days)	Total Expense per Patient Day
1935	0.7	174.2	257.4	
1945	0.7	164.7	253.1	
1955	0.7	145.9	218.9	$7.20
1965	0.3	52.4	182.5	$16.70
1966	0.2	39.9	168.3	$18.27

APPENDIX

TABLE 24

DEATH RATE FOR THE 10 LEADING CAUSES OF DEATH, 1966[116]

(Rate per 100,000 Population)

Disease	Death Rate
Diseases of the heart	371.2
Malignant neoplasms	155.1
Vascular diseases affecting central nervous system	104.6
Accidents	58.0
Influenza and pneumonia	32.5
Certain diseases of early infancy	26.4
General arteriosclerosis	19.9
Diabetes mellitus	17.7
Other diseases of the circulatory system	14.6
Other bronchiopneumonic diseases	14.5

TABLE 25

DEATH RATE (1950 to 1966) AND DEATHS (1965 AND 1966) FROM SELECTED CAUSES[116]

	Deaths per 100,000 Population						Total Deaths	
	1950	1955	1960	1964	1965	1966	1965	1966
All causes*	963.8	930.4	954.7	939.6	943.2	951.3	1,828,136	1,863,149
Tuberculosis (all forms)	22.5	9.1	6.1	4.3	4.1	3.9	7,934	7,625
Meningococcal infection	0.6	0.6	0.4	0.4	0.5	0.4	850	876
Asthma	2.9	3.6	3.0	2.3	2.3	2.2	4,520	4,324
Influenza and pneumonia (except pneumonia of newborn)	31.3	27.1	37.3	31.1	31.9	32.5	61,903	63,615
Influenza	4.4	1.7	4.4	0.9	1.2	1.4	2,295	2,830
Pneumonia	26.9	25.4	32.9	30.2	30.8	31.0	59,608	60,785
Bronchitis	2.0	1.9	2.4	2.8	3.0	3.1	5,772	6,151

*All causes listed in the complete table.

TABLE 26

SPECIFIED REPORTABLE DISEASES: CASES REPORTED,* 1945-1966[116]

Disease	1945	1950	1955	1960	1963	1964	1965	1966
Diphtheria	18,675	5,796	1,984	918	314	293	164	209
Measles	146,013	319,124	555,156	441,703	385,156	458,083	261,904	204,136
Meningococcal infection	8,208	3,788	3,455	2,259	2,470	2,826	3,040	3,381
Pertussis (whooping cough)	133,792	120,718	62,786	14,809	17,135	13,005	6,799	7,717
Psittacosis	27	26	334	113	76	53	60	50
Streptococcal sore throat and scarlet fever	185,570	66,494	147,502	315,173	342,161	404,334	395,168	427,752
Tuberculosis (newly reported active cases)			76,245	55,494	54,062	50,874	49,016	47,767

*Figures should be interpreted with caution. Reporting of some of these diseases is known to be incomplete and only indicates trends of disease incidence.

APPENDIX

TABLE 27

RESPIRATORY DISEASES IN THE UNITED STATES, JULY 1966-JUNE 1967[117]

Incidence and Effects	Common Cold	Influenza	Pneumonia	Bronchitis
Incidence (x 1000)	109,713	55,382	2,013	3,411
Days of restricted activity (x 1000)	263,622	186,514	26,409	19,966
Days of bed disability (x 1000)	109,999	102,016	16,406	10,392

APPENDIX

TABLE 28

AGE-SPECIFIC DISEASE RATES PER 100,000 POPULATION PER YEAR, 1959-61[28]

Cause	Rate per Age Group										
	0.4	4-14	15-24	25-34	35-44	45-54	55-64	65-74	75-84	85+	All
Tuberculosis, respiratory	0.22	0.03	0.41	2.18	5.05	9.70	16.00	23.03	31.88	36.70	5.89
Meningococcal infections	1.90	0.20	0.16	0.06	0.06	0.16	0.14	0.19	0.23	0.19	0.34
Asthma	0.39	0.22	0.39	0.81	1.63	3.44	7.05	12.29	16.91	16.08	2.69
Influenza and pneumonia (except of newborn)	52.95	2.27	2.46	4.12	8.70	18.12	38.81	101.93	315.73	998.74	32.07
Acute bronchitis	2.84	0.14	0.09	0.08	0.16	0.35	0.65	1.10	2.25	7.40	0.64
Chronic and Unqualified bronchitis	1.60	0.08	0.08	0.08	0.29	1.06	3.56	8.57	12.79	12.68	1.63
Age distribution for a standard million population	113,320	197,773	133,948	127,247	134,290	114,238	86,839	61,324	25,839	5,182	